	Type	L #	Hits	Search Text	DBs	Time Stamp	Comments	Error Definition	Errors
1	BRS	L1	427	amyloid adj fibril	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/09/25 12:50			0
2	BRS	L2	40255	immune adj response	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/09/25 12:52			0
3	BRS	L3	12	1 same 2	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/09/25 13:06			0
4	BRS	L4	17246	(immunoglobulin adj light adj chain) or (amyloid adj A adj protein) or (beta adj 2-microglobulin) or transthyretin or (cystatin adj C) or gelsolin or procalcitonin or (prp adj protein) or (amyloid adj beta-protein) or (apoA adj (amyloid adj fibril)) or lysozyme	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/09/25 13:06			0
5	BRS	L5	97	1 same 4	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/09/25 13:06			0
6	BRS	L6	1	5 same 2	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/09/25 13:07			0
7	BRS	L7	61	1 same synthetic	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/09/25 13:07			0
8	BRS	L8	13	1 same recombinant	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/09/25 13:07			0

	Type	L #	Hits	Search Text	DBs	Time Stamp	Comments	Error Definition	Errors
9	BRS	L9	35	1 same homologous	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/09/25 13:08			0
10	BRS	L10	1	1 same heterologous	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/09/25 13:08			0
11	BRS	L11	4	(7 or 8 or 9 or 10) same 2	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/09/25 13:09			0
12	BRS	L12	45468	vaccine	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/09/25 13:09			0
13	BRS	L13	4	12 same 1	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/09/25 13:10			0
14	BRS	L14	81619	adjuvant	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/09/25 13:13			0
15	BRS	L15	2	13 same 14	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/09/25 13:13			0

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FILE 'MEDLINE' ENTERED AT 13:19:3[redacted]N 25 SEP 2003

FILE 'CAPLUS' ENTERED AT 13:19:33 ON 25 SEP 2003
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
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FILE 'SCISEARCH' ENTERED AT 13:19:33 ON 25 SEP 2003
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FILE 'AGRICOLA' ENTERED AT 13:19:33 ON 25 SEP 2003

=> s amyloid fibril
L1         9106 AMYLOID FIBRIL

=> s immune response
L2       378632 IMMUNE RESPONSE

=> s (immunoglobin light chain) or (amyloid A protein) or (beta 2-microglobulin) or transthyretin
   4 FILES SEARCHED...
L3        55029 (IMMUNOGLOBIN LIGHT CHAIN) OR (AMYLOID A PROTEIN) OR (BETA 2-MIC
                ROGLOBULIN) OR TRANSTHYRETIN OR (CYSTATIN C) OR GELSOLIN OR
                PROCLACITONIN OR (PRP PROTEIN)

=> s (amyloid beta-protein) or (apoA 1) or lysozyme
L4       103850 (AMYLOID BETA-PROTEIN) OR (APOA 1) OR LYSOZYME

=> s l1 (p) l2
L5           16 L1 (P) L2

=> duplicate remove l5
DUPLICATE PREFERENCE IS 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH'
KEEP DUPLICATES FROM MORE THAN ONE FILE?  Y/(N):n
PROCESSING COMPLETED FOR L5
L6             6 DUPLICATE REMOVE L5 (10 DUPLICATES REMOVED)

=> d l6 1-6 ibib abs

L6   ANSWER 1 OF 6     MEDLINE on STN                    DUPLICATE 1
ACCESSION NUMBER:   2003379971     IN-PROCESS
DOCUMENT NUMBER:    22797269   PubMed ID: 12914815
TITLE:              "Eat me" and "don't eat me" signals govern the innate
                    immune response and tissue repair in the CNS: emphasis on
                    the critical role of the complement system.
AUTHOR:             Elward Kristina; Gasque Philippe
CORPORATE SOURCE:   Brain Inflammation and Immunity Group (BIIG), Department of
                    Medical Biochemistry and Immunology, University of Wales
                    College of Medicine, Cardiff CF144XN, Wales, UK.
SOURCE:             MOLECULAR IMMUNOLOGY, (2003 Sep) 40 (2-4) 85-94.
                    Journal code: 7905289. ISSN: 0161-5890.
PUB. COUNTRY:       England: United Kingdom
DOCUMENT TYPE:      Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:           English
FILE SEGMENT:       IN-PROCESS; NONINDEXED; Priority Journals
ENTRY DATE:         Entered STN: 20030814
                    Last Updated on STN: 20030913
AB   A full innate immune system (e.g. complement system, scavenger receptors,
     Toll-like receptors (TLR)) has been described in the CNS and is thought to
     be an extremely efficient army designed to fight against invading
     pathogens and toxic cell debris such as apoptotic cells and
       ***amyloid***     ***fibrils***  . The binding of soluble or secreted
     innate immune molecules on pathogen-associated molecular patterns (PAMPs)
     as well as apoptotic cell-associated molecular patterns (ACAMPs) provide
     several "eat me" signals to promote the safe disposal of the intruders by
     professional and amateur phagocytes. These patterns are deciphered by
     receptors (pattern recognition receptors, PRRs; e.g. CR3) that control
     phagocytosis and associated inflammatory response depending on the meaning
     of these signals. Importantly, in order to avoid excessive collateral
     damage of surrounding cells, it is increasingly evident that "don't eat
     me" signals (coined herein as self-associated molecular patterns, SAMPs;
```

e.g. complement regulatory proteins, CD200) are of paramount importance to signal a robust anti-inflammatory response and promote tissue repair. Further knowledge of the innate ***immune*** ***response*** in the CNS will greatly help to delineate the novel therapeutic routes to protect from CNS inflammation and neurodegeneration.

L6 ANSWER 2 OF 6 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 2002632808 MEDLINE
DOCUMENT NUMBER: 22278745 PubMed ID: 12391599
TITLE: Alzheimer's disease with spastic paresis and cotton wool type plaques.
AUTHOR: Tabira Takeshi; Chui De Hua; Nakayama Hiroshi; Kuroda Shigetoshi; Shibuya Makoto
CORPORATE SOURCE: National Institute for Longevity Sciences, Obu, Aichi, Japan.. tabira@nils.go.jp
SOURCE: JOURNAL OF NEUROSCIENCE RESEARCH, (2002 Nov 1) 70 (3) 367-72. Ref: 21
Journal code: 7600111. ISSN: 0360-4012.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW OF REPORTED CASES)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200212
ENTRY DATE: Entered STN: 20021023
Last Updated on STN: 20021218
Entered Medline: 20021213

AB We reviewed Alzheimer's cases with spastic paresis and cotton wool type plaques in five Japanese and nine Caucasian cases. Most were early onset familial Alzheimer's disease with presenilin 1 mutations. The cotton wool type plaques were related to extremely high production of A beta 42, due mainly to presenilin 1 mutations and low ***immune*** ***responses*** . Cotton wool plaques were numerous in the entire central nervous system, including basal ganglia, brainstem and even in spinal cord. Cotton wool type plaques were composed of slightly electron dense synaptic structures, but ***amyloid*** ***fibrils*** were rarely found. Such a high accumulation of A beta 42 may cause degeneration of the pyramidal tract and basal ganglia from an early stage of Alzheimer's disease.
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L6 ANSWER 3 OF 6 MEDLINE on STN DUPLICATE 3
ACCESSION NUMBER: 2001654245 MEDLINE
DOCUMENT NUMBER: 21558631 PubMed ID: 11701763
TITLE: Vaccination with soluble Abeta oligomers generates toxicity-neutralizing antibodies.
AUTHOR: Lambert M P; Viola K L; Chromy B A; Chang L; Morgan T E; Yu J; Venton D L; Krafft G A; Finch C E; Klein W L
CORPORATE SOURCE: Department of Neurobiology and Physiology, Northwestern University, Evanston, IL 60208, USA.
CONTRACT NUMBER: AG 13499 (NIA)
PO1 AG13138 (NIA)
SOURCE: JOURNAL OF NEUROCHEMISTRY, (2001 Nov) 79 (3) 595-605.
Journal code: 2985190R. ISSN: 0022-3042.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200112
ENTRY DATE: Entered STN: 20011115
Last Updated on STN: 20020123
Entered Medline: 20011207

AB In recent studies of transgenic models of Alzheimer's disease (AD), it has been reported that antibodies to aged beta amyloid peptide 1-42 (Abeta(1-42)) solutions (mixtures of Abeta monomers, oligomers and ***amyloid*** ***fibrils***) cause conspicuous reduction of amyloid plaques and neurological improvement. In some cases, however, neurological improvement has been independent of obvious plaque reduction, and it has been suggested that immunization might neutralize soluble, non-fibrillar forms of Abeta. It is now known that Abeta toxicity resides not only in fibrils, but also in soluble protofibrils and oligomers. The current study has investigated the ***immune*** ***response*** to low doses of Abeta(1-42) oligomers and the characteristics of the antibodies they induce. Rabbits that were injected with Abeta(1-42) solutions containing only monomers and oligomers produced antibodies that preferentially bound to assembled forms of Abeta in immunoblots and in

physiological solutions. The antibodies have proven useful for assays that can detect inhibitors of oligomer formation, for immunofluorescence localization of cell-attached oligomers to receptor-like puncta, and for immunoblots that show the presence of SDS-stable oligomers in Alzheimer's brain tissue. The antibodies, moreover, were found to neutralize the toxicity of soluble oligomers in cell culture. Results support the hypothesis that immunizations of transgenic mice derive therapeutic benefit from the immuno-neutralization of soluble Abeta-derived toxins. Analogous immuno-neutralization of oligomers in humans may be a key in AD vaccines.

L6 ANSWER 4 OF 6 CAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 1999:753260 CAPLUS
DOCUMENT NUMBER: 131:350268
TITLE: Amyloid removal using anti-amyloid antibodies
INVENTOR(S): Solomon, Alan; Hrncic, Rudi; Wall, Jonathan S.
PATENT ASSIGNEE(S): The University of Tennessee Research Corporation, USA
SOURCE: PCT Int. Appl., 34 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9960024	A1	19991125	WO 1999-US11200	19990521
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2325600	AA	19991125	CA 1999-2325600	19990521
AU 9940075	A1	19991206	AU 1999-40075	19990521
EP 1078005	A1	20010228	EP 1999-923260	19990521
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
JP 2002515235	T2	20020528	JP 2000-549642	19990521
US 2003147882	A1	20030807	US 1999-316387	19990521
ZA 2000007811	A	20020621	ZA 2000-7811	20001221

PRIORITY APPLN. INFO.: US 1998-86198P P 19980521
WO 1999-US11200 W 19990521

AB The authors disclose that the cell-mediated ***immune*** ***response*** to deposits of ***amyloid*** ***fibrils*** is enhanced by the opsonizing activity of anti-amyloid antibodies. In one example, amyloid deposits were shown to resolved in mice given anti-light chain antibodies; resoln. was myeloid cell (CD18)-dependent.
REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 5 OF 6 MEDLINE on STN
ACCESSION NUMBER: 88312016 MEDLINE
DOCUMENT NUMBER: 88312016 PubMed ID: 3044707
TITLE: Neuropathology of unconventional virus infections: molecular pathology of spongiform change and amyloid plaque deposition.
AUTHOR: Masters C L; Beyreuther K
CORPORATE SOURCE: Department of Pathology, University of Western Australia, Perth.
SOURCE: CIBA FOUNDATION SYMPOSIUM, (1988) 135 24-36. Ref: 28
Journal code: 0356636. ISSN: 0300-5208.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198809
ENTRY DATE: Entered STN: 19900308
Last Updated on STN: 19980206
Entered Medline: 19880927

AB To the triad of neuronal loss, gliosis and spongiform change as characteristic morphological changes associated with infection of the

central nervous system, one can now add the presence of scrapie-associated filaments (SAF)/PrP rods. While the host's ***immune*** ***response*** is conspicuous by its absence, the vigorous astrocytic response is presumptive evidence of the host's ability to recognize and respond to the primary neuronal insult. We assume that the spongiform change and vacuolation of neurons are of fundamental importance in the pathogenesis of the disease, realizing that neither is specific or essential for the replication of the infectious agent. The topographical distribution of lesions is partly explained by the portal of entry and retrograde spread of the virus. The temporal progression of the lesions is more clearly determined by the host genes, best illustrated by studies of the incubation period. The molecular basis of the spongiform change is unknown but it is presumed to involve some disturbance of membrane metabolism. The recognition of PrP as a membrane glycoprotein invites proposals for its role in the development of these spongiform lesions. Extracellular amyloid occurs as plaques or congophilic angiopathy in some instances, and provides the best evidence that Alzheimer's disease (AD) is in some way related to the unconventional virus diseases. However, the protein subunit (A4) of the ***amyloid*** ***fibril*** in AD and its precursor are quite distinct from the PrP subunit which constitutes the ***amyloid*** ***fibril*** in these infectious diseases. It is still unclear whether the PrP subunit in the SAF has exactly the same composition as in the extracellular ***amyloid*** ***fibril*** . Our results suggest that only a fragment of the PrP molecule is the major constituent of the extracellular fibril. Since both PrP and A4 are derived from membrane glycoproteins, the elucidation of their normal function is likely to lead to a better understanding of the spongiform and amyloidogenic lesions in these diseases.

L6 ANSWER 6 OF 6 EMBASE COPYRIGHT 2003 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN

ACCESSION NUMBER: 80091190 EMBASE
DOCUMENT NUMBER: 1980091190
TITLE: [Pathological immunology of amyloidosis].
IMMUNOPATOLOGIA DELL'AMILOIDOSI.
AUTHOR: Clerici E.
CORPORATE SOURCE: Catt. Immunol., Univ. Studi, Milano, Italy
SOURCE: Giornale di Gerontologia, (1979) 27/9 (577-582).
CODEN: GIGEAU
COUNTRY: Italy
DOCUMENT TYPE: Journal
FILE SEGMENT: 020 Gerontology and Geriatrics
005 General Pathology and Pathological Anatomy
026 Immunology, Serology and Transplantation
LANGUAGE: Italian
SUMMARY LANGUAGE: English

AB Amyloidosis has as it distinguishing feature deposits of antiparallel .beta.-pleated sheet fibrils which are responsible for the pathologic manifestations of the disease. In a group of cases the protein of the fibrils is mainly composed by light polypeptide chain and/or its amino-terminal fragment. In another group of cases the major fibril protein is of a yet unknown origin. Often, if not invariably, an immunoglobulin protein is also found in these cases. During the experimental casein amyloidosis in mice, the percentage of B-lymphocytes and the macrophages of the spleen increases, while that of T-lymphocyts significantly decreases as compared to controls. Contemporaneously to these cellular modifications, both the in vivo and in vitro ***immune*** ***response*** to foreign antigens is sharply reduced, as compared to that of the normal counterparts. It is suggested that such cellular and functional alterations are compatible with a sterile blastogenesis and with an aspecific hyperproduction of immunoglobulin light chains or immunoglobulin-related polypeptides which are either transformed or incorporated into ***amyloid*** ***fibrils*** .

=> d his

(FILE 'HOME' ENTERED AT 13:19:07 ON 25 SEP 2003)

FILE 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH, AGRICOLA' ENTERED AT 13:19:33 ON 25 SEP 2003
L1 9106 S AMYLOID FIBRIL
L2 378632 S IMMUNE RESPONSE
L3 55029 S (IMMUNOGLOBIN LIGHT CHAIN) OR (AMYLOID A PROTEIN) OR (BETA 2-
L4 103850 S (AMYLOID BETA-PROTEIN) OR (APOA 1) OR LYSOZYME
L5 16 S L1 (P) L2
L6 6 DUPLICATE REMOVE L5 (10 DUPLICATES REMOVED)

```
=> s l1 (p) (l3 or l4)
L7        2050 L1 (P) (L3 OR L4)

=> s l2 (p) l7
L8           0 L2 (P) L7

=> s l6 (p) remov? (p) (amyloid deposit)
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L55 (P) REMOV?'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'REMOV? (P) '
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L59 (P) REMOV?'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'REMOV? (P) '
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L61 (P) REMOV?'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'REMOV? (P) '
L9           0 L6 (P) REMOV? (P) (AMYLOID DEPOSIT)

=> s l1 (p) (synthetic or recombinant or homologous or hetrologous)
L10       1005 L1 (P) (SYNTHETIC OR RECOMBINANT OR HOMOLOGOUS OR HETROLOGOUS)

=> s vaccine
L11     399015 VACCINE

=> s l11 (p) l1
L12          21 L11 (P) L1

=> s l12 (p) l2
L13           5 L12 (P) L2

=> duplicate remove l13
DUPLICATE PREFERENCE IS 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH'
KEEP DUPLICATES FROM MORE THAN ONE FILE?  Y/(N):n
PROCESSING COMPLETED FOR L13
L14           1 DUPLICATE REMOVE L13 (4 DUPLICATES REMOVED)

=> d l14 1 ibib abs

L14  ANSWER 1 OF 1     MEDLINE on STN                    DUPLICATE 1
ACCESSION NUMBER:    2001654245     MEDLINE
DOCUMENT NUMBER:     21558631   PubMed ID: 11701763
TITLE:               Vaccination with soluble Abeta oligomers generates
                     toxicity-neutralizing antibodies.
AUTHOR:              Lambert M P; Viola K L; Chromy B A; Chang L; Morgan T E; Yu
                     J; Venton D L; Krafft G A; Finch C E; Klein W L
CORPORATE SOURCE:    Department of Neurobiology and Physiology, Northwestern
                     University, Evanston, IL 60208, USA.
CONTRACT NUMBER:     AG 13499 (NIA)
     PO1 AG13138 (NIA)
SOURCE:              JOURNAL OF NEUROCHEMISTRY, (2001 Nov) 79 (3) 595-605.
                     Journal code: 2985190R. ISSN: 0022-3042.
PUB. COUNTRY:        United States
DOCUMENT TYPE:       Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:            English
FILE SEGMENT:        Priority Journals
ENTRY MONTH:         200112
ENTRY DATE:          Entered STN: 20011115
                     Last Updated on STN: 20020123
                     Entered Medline: 20011207
AB   In recent studies of transgenic models of Alzheimer's disease (AD), it has
     been reported that antibodies to aged beta amyloid peptide 1-42
     (Abeta(1-42)) solutions (mixtures of Abeta monomers, oligomers and
       ***amyloid***     ***fibrils***  ) cause conspicuous reduction of amyloid
     plaques and neurological improvement.  In some cases, however,
     neurological improvement has been independent of obvious plaque reduction,
     and it has been suggested that immunization might neutralize soluble,
     non-fibrillar forms of Abeta.  It is now known that Abeta toxicity resides
     not only in fibrils, but also in soluble protofibrils and oligomers.  The
     current study has investigated the   ***immune***     ***response***   to
     low doses of Abeta(1-42) oligomers and the characteristics of the
     antibodies they induce.  Rabbits that were injected with Abeta(1-42)
     solutions containing only monomers and oligomers produced antibodies that
     preferentially bound to assembled forms of Abeta in immunoblots and in
```

physiological solutions. The antibodies have proven useful for assays that can detect inhibitors of oligomer formation, for immunofluorescence localization of cell-attached oligomers to receptor-like puncta, and for immunoblots that show the presence of SDS-stable oligomers in Alzheimer's brain tissue. The antibodies, moreover, were found to neutralize the toxicity of soluble oligomers in cell culture. Results support the hypothesis that immunizations of transgenic mice derive therapeutic benefit from the immuno-neutralization of soluble Abeta-derived toxins. Analogous immuno-neutralization of oligomers in humans may be a key in AD ***vaccines*** .

```
=> s adjuvant
L15     230166 ADJUVANT

=> d his

     (FILE 'HOME' ENTERED AT 13:19:07 ON 25 SEP 2003)

     FILE 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH, AGRICOLA' ENTERED AT
     13:19:33 ON 25 SEP 2003
L1        9106 S AMYLOID FIBRIL
L2      378632 S IMMUNE RESPONSE
L3       55029 S (IMMUNOGLOBIN LIGHT CHAIN) OR (AMYLOID A PROTEIN) OR (BETA 2-
L4      103850 S (AMYLOID BETA-PROTEIN) OR (APOA 1) OR LYSOZYME
L5          16 S L1 (P) L2
L6           6 DUPLICATE REMOVE L5 (10 DUPLICATES REMOVED)
L7        2050 S L1 (P) (L3 OR L4)
L8           0 S L2 (P) L7
L9           0 S L6 (P) REMOV? (P) (AMYLOID DEPOSIT)
L10       1005 S L1 (P) (SYNTHETIC OR RECOMBINANT OR HOMOLOGOUS OR HETROLOGOUS
L11     399015 S VACCINE
L12         21 S L11 (P) L1
L13          5 S L12 (P) L2
L14          1 DUPLICATE REMOVE L13 (4 DUPLICATES REMOVED)
L15     230166 S ADJUVANT

=> s l12 (p) l15
L16          8 L12 (P) L15

=> s l16 (p) l2
L17          0 L16 (P) L2

=> d his

     (FILE 'HOME' ENTERED AT 13:19:07 ON 25 SEP 2003)

     FILE 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH, AGRICOLA' ENTERED AT
     13:19:33 ON 25 SEP 2003
L1        9106 S AMYLOID FIBRIL
L2      378632 S IMMUNE RESPONSE
L3       55029 S (IMMUNOGLOBIN LIGHT CHAIN) OR (AMYLOID A PROTEIN) OR (BETA 2-
L4      103850 S (AMYLOID BETA-PROTEIN) OR (APOA 1) OR LYSOZYME
L5          16 S L1 (P) L2
L6           6 DUPLICATE REMOVE L5 (10 DUPLICATES REMOVED)
L7        2050 S L1 (P) (L3 OR L4)
L8           0 S L2 (P) L7
L9           0 S L6 (P) REMOV? (P) (AMYLOID DEPOSIT)
L10       1005 S L1 (P) (SYNTHETIC OR RECOMBINANT OR HOMOLOGOUS OR HETROLOGOUS
L11     399015 S VACCINE
L12         21 S L11 (P) L1
L13          5 S L12 (P) L2
L14          1 DUPLICATE REMOVE L13 (4 DUPLICATES REMOVED)
L15     230166 S ADJUVANT
L16          8 S L12 (P) L15
L17          0 S L16 (P) L2

=> s solomon alan/au
L18        173 SOLOMON ALAN/AU

=> s wall jonathan/au
L19         15 WALL JONATHAN/AU

=> s hrncic rudi/au
L20         14 HRNCIC RUDI/AU

=> s schell maria/au
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L21         19 SCHELL MARIA/AU

=> s (l18 or l19 or l20 or l21)
L22        176 (L18 OR L19 OR L20 OR L21)

=> s l22 and l6
L23          1 L22 AND L6

=> d l23 1 ibib abs

L23  ANSWER 1 OF 1  CAPLUS  COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER:         1999:753260  CAPLUS
DOCUMENT NUMBER:          131:350268
TITLE:                    Amyloid removal using anti-amyloid antibodies
INVENTOR(S):                ***Solomon, Alan***  ;   ***Hrncic, Rudi***  ; Wall,
                          Jonathan S.
PATENT ASSIGNEE(S):       The University of Tennessee Research Corporation, USA
SOURCE:                   PCT Int. Appl., 34 pp.
                          CODEN: PIXXD2
DOCUMENT TYPE:            Patent
LANGUAGE:                 English
FAMILY ACC. NUM. COUNT:   1
PATENT INFORMATION:

     PATENT NO.        KIND  DATE           APPLICATION NO.  DATE
     ---------------   ----  --------       ---------------  --------
     WO 9960024         A1   19991125       WO 1999-US11200  19990521
         W:  AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ,
             DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS,
             JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK,
             MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ,
             TM, TR, TT, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD,
             RU, TJ, TM
         RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK,
             ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG,
             CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
     CA 2325600         AA   19991125       CA 1999-2325600  19990521
     AU 9940075         A1   19991206       AU 1999-40075    19990521
     EP 1078005         A1   20010228       EP 1999-923260   19990521
         R:  AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
             IE, SI, LT, LV, FI, RO
     JP 2002515235      T2   20020528       JP 2000-549642   19990521
     US 2003147882      A1   20030807       US 1999-316387   19990521
     ZA 2000007811      A    20020621       ZA 2000-7811     20001221
PRIORITY APPLN. INFO.:                    US 1998-86198P   P  19980521
                                          WO 1999-US11200  W  19990521
AB   The authors disclose that the cell-mediated   ***immune***
       ***response***   to deposits of   ***amyloid***     ***fibrils***   is
     enhanced by the opsonizing activity of anti-amyloid antibodies.  In one
     example, amyloid deposits were shown to resolved in mice given anti-light
     chain antibodies; resoln. was myeloid cell (CD18)-dependent.
REFERENCE COUNT:          4     THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS
                                RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT


=> d his

     (FILE 'HOME' ENTERED AT 13:19:07 ON 25 SEP 2003)

     FILE 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH, AGRICOLA' ENTERED AT
     13:19:33 ON 25 SEP 2003
L1          9106 S AMYLOID FIBRIL
L2        378632 S IMMUNE RESPONSE
L3         55029 S (IMMUNOGLOBIN LIGHT CHAIN) OR (AMYLOID A PROTEIN) OR (BETA 2-
L4        103850 S (AMYLOID BETA-PROTEIN) OR (APOA 1) OR LYSOZYME
L5            16 S L1 (P) L2
L6             6 DUPLICATE REMOVE L5 (10 DUPLICATES REMOVED)
L7          2050 S L1 (P) (L3 OR L4)
L8             0 S L2 (P) L7
L9             0 S L6 (P) REMOV? (P) (AMYLOID DEPOSIT)
L10         1005 S L1 (P) (SYNTHETIC OR RECOMBINANT OR HOMOLOGOUS OR HETROLOGOUS
L11       399015 S VACCINE
L12           21 S L11 (P) L1
L13            5 S L12 (P) L2
L14            1 DUPLICATE REMOVE L13 (4 DUPLICATES REMOVED)
L15       230166 S ADJUVANT
L16            8 S L12 (P) L15
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L17           0 S L16 (P) L2
L18         173 S SOLOMON ALAN/AU
L19          15 S WALL JONATHAN/AU
L20          14 S HRNCIC RUDI/AU
L21          19 S SCHELL MARIA/AU
L22         176 S (L18 OR L19 OR L20 OR L21)
L23           1 S L22 AND L6

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                                                      ENTRY    SESSION
FULL ESTIMATED COST                                   95.95      96.16

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)          SINCE FILE      TOTAL
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CA SUBSCRIBER PRICE                                   -1.30      -1.30

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